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L7 and L5	72

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JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L8

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Search History

DATE: Friday, April 15, 2005 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L8	L7 and l5	72	L8
L7	(filamin 1) or (filamin a)	7143934	L7
L6	(filamin 1) or (filamin a)	7143934	L6
L5	L4 and l3	72	L5
L4	nagano.in.	1759	L4
L3	sato.in.	16650	L3
L2	6569662.pn.	1	L2
L1	5599919.pn.	1	L1

END OF SEARCH HISTORY

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 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  FEB 25   CA/CAPLUS - Russian Agency for Patents and Trademarks
                   (ROSPATENT) added to list of core patent offices covered
 NEWS  4  FEB 28   PATDPAFULL - New display fields provide for legal status
                   data from INPADOC
 NEWS  5  FEB 28   BABS - Current-awareness alerts (SDIs) available
 NEWS  6  FEB 28   MEDLINE/LMEDLINE reloaded
 NEWS  7  MAR 02   GBFULL: New full-text patent database on STN
 NEWS  8  MAR 03   REGISTRY/ZREGISTRY - Sequence annotations enhanced
 NEWS  9  MAR 03   MEDLINE file segment of TOXCENTER reloaded
 NEWS  10 MAR 22   KOREAPAT now updated monthly; patent information enhanced
 NEWS  11 MAR 22   Original IDE display format returns to REGISTRY/ZREGISTRY
 NEWS  12 MAR 22   PATDPASPC - New patent database available
 NEWS  13 MAR 22   REGISTRY/ZREGISTRY enhanced with experimental property tags
 NEWS  14 APR 04   EPFULL enhanced with additional patent information and new
                   fields
 NEWS  15 APR 04   EMBASE - Database reloaded and enhanced

 NEWS EXPRESS  JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
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FILE 'HOME' ENTERED AT 16:42:09 ON 15 APR 2005

=> file medline, uspatful, dgene, embase, wpids, fsta, jicst
COST IN U.S. DOLLARS                        SINCE FILE      TOTAL
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FILE 'MEDLINE' ENTERED AT 16:42:29 ON 15 APR 2005

FILE 'USPATFULL' ENTERED AT 16:42:29 ON 15 APR 2005
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=> s filamin A or filamin 1
   3 FILES SEARCHED...
L1   249 FILAMIN A OR FILAMIN 1

=> s l1 and (cell migration or cell death control)
   2 FILES SEARCHED...
   3 FILES SEARCHED...
   4 FILES SEARCHED...
L2   39 L1 AND (CELL MIGRATION OR CELL DEATH CONTROL)

=> s l2 and (fragment or variant or subsitution or deletion or addition)
L3   14 L2 AND (FRAGMENT OR VARIANT OR SUBSITUTION OR DELETION OR ADDIT
              ION)

=> d l14 ti abs ibib tot
L14 NOT FOUND
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has been deleted. To see the L-numbers currently defined in this
session, enter DISPLAY HISTORY at an arrow prompt (=>).

=> d l3 ti abs ibib tot

L3   ANSWER 1 OF 14   MEDLINE on STN
TI   **Cell migration** and cerebral cortical development.
AB   This annotation describes the clinical and pathological features of
     several conditions believed to result from a primary defect in
     cell migration which include the lissencephalies,
     pachygria, polymicrogyrias, and focal cortical dysplasia. A variety of
     factors must be considered in pathogeneses, including cellular
     proliferation, cell death, post-migrational intracortical growth and
     development, axonogenesis and dendritogenesis. At least two distinct
     types of lissencephaly exist. Classic (also known as Type I)
     lissencephaly is the prototypic pattern being seen in autosomal dominant
     Miller-Dieker syndrome, in **addition** to autosomal recessive and
     X-linked forms. The Miller-Dieker syndrome locus (LIS-1) encodes the
     platelet activating factor acetylhydrolase-1, beta1 subunit. The gene for
     an X-linked form of lissencephaly (XLIS) encodes a protein called
     doublecortin. Cobblestone (type II) lissencephaly is most commonly seen
     in patients with the Walker-Warburg syndrome, and also occurs in a group
     of disorders associated with congenital muscular dystrophy, including
     Finnish 'muscle-eye-brain' disease and Fukuyama muscular dystrophy.
     Controversy exits as to whether polymicrogyria is a malformation or a
     disruption of development. The answer is likely both. Polymicrogyria is
     believed to arise from defects occurring between 17 and 25 or 26 weeks
     gestation. Heterotopia can be sporadic, inherited as a simple Mendelian
     trait, or may be part of a more complex syndrome being characterized by
     collections of disorganized grey matter in inappropriate places. X-linked
     periventricular heterotopia syndrome is caused by mutations in
     filamin-1. In **addition** to those described
     above, many other syndromes show lissencephaly, pachygyria and
     polymicrogyria and many cases are not easily classified into any
     particular syndrome.
ACCESSION NUMBER: 2001209709   MEDLINE

DOCUMENT NUMBER: PubMed ID: 11298998
TITLE: **Cell migration** and cerebral cortical development.
AUTHOR: Golden J A
CORPORATE SOURCE: Department of Pathology, The Children's Hospital of Philadelphia and the University of Pennsylvania School of Medicine, Philadelphia, PA 19104, USA..
goldenj@mail.med.upenn.edu
SOURCE: Neuropathology and applied neurobiology, (2001 Feb) 27 (1) 22-8. Ref: 35
Journal code: 7609829. ISSN: 0305-1846.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

L3 ANSWER 2 OF 14 USPATFULL on STN
TI Specific markers for diabetes
AB The present invention provides polypeptides which are correlated with pre-diabetes, diabetes or susceptibility to diabetes which can be used as markers for diagnosis of pre-diabetes, diabetes or a susceptibility or predisposition to develop diabetes. The invention also provides methods for the diagnosis of pre-diabetes, diabetes and/or the susceptibility to diabetes by obtaining a biological sample and detecting and/or measuring the increase of one or more polypeptides as disclosed herein. Screening methods relating to agonists and antagonists of the specific polypeptides disclosed herein are provided. Antibodies may also be raised against these polypeptide markers for the detection and/or treatment of diabetes. Proteins, protein fragments or peptides can be used for the treatment of diabetes or pre-diabetes.

ACCESSION NUMBER: 2005:87343 USPATFULL
TITLE: Specific markers for diabetes
INVENTOR(S): Kochan, Jarema Peter, Towaco, NJ, UNITED STATES
Martin, Mitchell Lee, Verona, NJ, UNITED STATES
Rosinski, James Andrew, Nutley, NJ, UNITED STATES
PATENT ASSIGNEE(S): Hoffmann-La Roche Inc., Nutley, NJ (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005074805	A1	20050407	
APPLICATION INFO.:	US 2004-952459	A1	20040928	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-508699P	20031003 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOFFMANN-LA ROCHE INC., PATENT LAW DEPARTMENT, 340 KINGSLAND STREET, NUTLEY, NJ, 07110
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 2961

L3 ANSWER 3 OF 14 USPATFULL on STN
TI Proteins having effects of controlling **cell migration** and cell death
AB The present invention relates to a protein having effects of controlling **cell migration** and cell death of such as neurons and a DNA encoding the protein, and an object of the present invention is to provide control **cell migration** and/or cell death and

a method for screening a promoter or an inhibitor of the effects of controlling **cell migration** and/or cell death with the use of proteins controlling the cell motility and cell death of neurons by interacting particularly with an actin-binding protein and promoting the degradation of the actin-binding protein and the DNA encoding the proteins. S-FILIP, L-FILIP and h-FILIP cDNAs, interacting with an actin-binding protein **Filamin 1**, and negatively controlling **cell migration** by promoting the degradation of the **Filamin 1**, and involved in the control of the cell death, were isolated and the full base sequences and amino acid sequences thereof were determined.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2004:292708 USPATFULL
TITLE: Proteins having effects of controlling **cell migration** and cell death
INVENTOR(S): Sato, Makoto, Fukui-shi, JAPAN
Nagano, Takashi, Sakai-gun, JAPAN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004229797	A1	20041118
APPLICATION INFO.:	US 2004-788793	A1	20040227 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2002-JP7676, filed on 29 Jul 2002, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	JP 2001-256910	20010827

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: THOMAS J. KOWALSKI, Esq., c/o FROMMER LAWRENCE & HAUG LLP, 745 Fifth Avenue, New York, NY, 10151
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 2861
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 4 OF 14 USPATFULL on STN
TI Specific markers for pancreatic cancer
AB The present invention provides polypeptides which are up- or down-regulated in pancreatic cancer and which can be used as markers for diagnosis of pancreatic cancer. The invention also provides an in vitro method for the diagnosis of pancreatic cancer and/or the susceptibility to pancreatic cancer comprising the steps of a) obtaining a biological sample; and b) detecting and/or measuring the increase of one or more polypeptides as disclosed herein. Furthermore, screening methods relating to inhibitors and antagonists of the specific polypeptides disclosed herein are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2004:280272 USPATFULL
TITLE: Specific markers for pancreatic cancer
INVENTOR(S): Chen, Jie, Beijing, CHINA
Hu, Liping, Beijing, CHINA
Liu, Tong Hua, Beijing, CHINA
Lu, Zhao Hui, Beijing, CHINA
Shen, Yan, Beijing, CHINA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004219572	A1	20041104
APPLICATION INFO.:	US 2003-733969	A1	20031211 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	EP 2002-28058	20021217

EP 2003-25237 20031105
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOFFMANN-LA ROCHE INC., PATENT LAW DEPARTMENT, 340 KINGSLAND STREET, NUTLEY, NJ, 07110
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
LINE COUNT: 8167
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 5 OF 14 USPATFULL on STN
TI Compositions and methods for prolonging survival of platelets
AB The present invention provides modified platelets having a reduced platelet clearance and methods for reducing platelet clearance. Also provided are compositions for the preservation of platelets. The invention also provides methods for making a pharmaceutical composition containing the modified platelets and for administering the pharmaceutical composition to a mammal to mediate hemostasis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:239232 USPATFULL
TITLE: Compositions and methods for prolonging survival of platelets
INVENTOR(S): Stossel, Thomas P., Belmont, MA, UNITED STATES
Hartwig, John H., Jamaica Plain, MA, UNITED STATES
Hoffmeister, Karin M., Cambridge, MA, UNITED STATES
Clausen, Henrik, Holte, DENMARK

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004185036	A1	20040923	
APPLICATION INFO.:	US 2003-704377	A1	20031107	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2002-424807P	20021108	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: John R. Van Amsterdam, Wolf, Greenfield & Sacks, P.C., 600 Atlantic Avenue, Boston, MA, 02210
NUMBER OF CLAIMS: 63
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Page(s)
LINE COUNT: 2465
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 6 OF 14 USPATFULL on STN
TI Modulators of angiogenesis
AB The present invention relates to regulation of angiogenesis. More particularly, the present invention is directed to nucleic acids encoding "angiogenesis regulatory proteins and nucleic acids" which are involved in modulation of angiogenesis. The invention further relates to methods for identifying and using agents, including small organic molecules, antibodies, peptides, cyclic peptides, nucleic acids, antisense nucleic acids, RNAi, and ribozymes, that modulate angiogenesis via modulation of angiogenesis regulatory proteins and nucleic acids; as well as to the use of expression profiles and compositions in diagnosis and therapy of diseases related to angiogenesis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:70006 USPATFULL
TITLE: Modulators of angiogenesis
INVENTOR(S): Lorens, James B., Portola Valley, CA, UNITED STATES
Xu, Weiduan, San Francisco, CA, UNITED STATES
Bogenberger, Jakob, San Francisco, CA, UNITED STATES
Holland, Sacha, San Francisco, CA, UNITED STATES
PATENT ASSIGNEE(S): Rigel Pharmaceuticals, Incorporated, South San Francisco, CA, UNITED STATES, 94080 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004053233	A1	20040318	
APPLICATION INFO.:	US 2002-231956	A1	20020830	(10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 37
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 2914
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 7 OF 14 USPATFULL on STN
TI GIPs, a family of polypeptides with transcription factor activity that interact with goodpasture antigen binding protein
AB The present invention provides isolated GPBP-interacting 90 and 130 kDa polypeptides, and portions thereof (GIP90/130 polypeptides), antibodies to the GIP90/130 polypeptides, and pharmaceutical compositions thereof. The present invention also provides isolated GIP90/130 nucleic acid sequences, expression vectors comprising the nucleic acid sequences, and host cells transfected with the expression vectors. The invention further provides methods for detecting the GIP90/130 polypeptides or nucleic acid sequences, methods for inhibiting interactions between GPBP and GIP90/130 polypeptides, between pol k76 and GIP90/130 polypeptides or aggregation of GIP90/130 polypeptides, and methods for treating patients with autoimmune disorders or cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:158941 USPATFULL
TITLE: GIPs, a family of polypeptides with transcription factor activity that interact with goodpasture antigen binding protein
INVENTOR(S): Saus, Juan, Valencia, SPAIN
Revert-Ros, Francisco, Valencia, SPAIN

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003108554	A1	20030612	
APPLICATION INFO.:	US 2002-309851	A1	20021204	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-338287P	20011207	(60)
	US 2002-382004P	20020520	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606
NUMBER OF CLAIMS: 33
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 3697
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 8 OF 14 USPATFULL on STN
TI Diagnosis and treatment of medical conditions associated with defective NFkappa B(NF-kappaB) activation
AB Incontinentia Pigmenti (IP) is a neurocutaneous genodermatosis that segregates as an X-linked dominant disorder with a high probability of prenatal male lethality. A locus in Xq28 containing NF-κB Essential Modulator, a gene product involved in the activation of NF-kB and central to many pro-inflammatory and apoptotic pathways, contains mutations in the majority of cases of IP. Disclosed are methods, compositions and kits directed to a defect in a NF-κB related disease such as IP.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:44745 USPATFULL
TITLE:	Diagnosis and treatment of medical conditions associated with defective NFkappa B(NF-kappaB) activation
INVENTOR(S):	Kenwrick, Sue J., Cambridge, UNITED KINGDOM Woffendin, Hayley, Cambridge, UNITED KINGDOM Munnich, Arnold, Paris, FRANCE Smahi, Asmae, Saint Ouen, FRANCE Israel, Alain, Paris, FRANCE Poustka, Annemarie, Heidelberg, GERMANY, FEDERAL REPUBLIC OF Heiss, Nina, Heidelberg, GERMANY, FEDERAL REPUBLIC OF D'Urso, Michele, Napoli, ITALY Lewis, Richard Alan, Houston, TX, UNITED STATES Nelson, David L., Houston, TX, UNITED STATES Aradhya, Swaroop, Houston, TX, UNITED STATES Levy, Moise, Houston, TX, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003032055	A1	20030213	
	US 6824972	B2	20041130	
APPLICATION INFO.:	US 2001-863049	A1	20010522	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-206223P	20000522 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	FULBRIGHT & JAWORSKI, LLP, 1301 MCKINNEY, SUITE 5100, HOUSTON, TX, 77010-3095
NUMBER OF CLAIMS:	49
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	13 Drawing Page(s)
LINE COUNT:	3161

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 9 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI **Filamin A** and FILIP (**Filamin A** -interacting protein) regulate cell polarity and motility in neocortical subventricular and intermediate zones during radial migration.

AB In the developing neocortex, most excitatory neurons are supplied and arranged through radial migration. Because neurons show global morphological changes and complicated behavior during that migration, precise regulation of cell shape and polarity is essential for proper migration and correct neocortical formation; however, how cell shape and polarity are regulated in migrating neuron remains elusive. We show here that **Filamin A**, a well known actin-binding protein, determines the shape of neocortical neurons during radial migration in vivo. Dysfunction of **Filamin A**, caused by a mutant **Filamin A** expression, prevents cells from acquiring consistent polarity toward specific direction and decreases motility in the subventricular and intermediate zones. In contrast, **Filamin A** overexpression, achieved by a short interfering RNA for **Filamin A**-interacting protein that induces **Filamin A** degradation (FILIP), promotes the development and maintenance of a bipolar shape also in the subventricular and intermediate zones. These results suggest that the amount of **Filamin A** helps migrating neurons determine their mode of migration, multipolar or bipolar, before entering the cortical plate and that FILIP is responsible, at least in part, for **Filamin A** content. In **addition**, our results also give a possible clue to understanding the pathogenesis of human malformation periventricular heterotopia, which is caused by various "loss-of-function" mutations in the **filamin A** gene.

ACCESSION NUMBER: 2004465578 EMBASE
TITLE: **Filamin A** and FILIP (**Filamin A**-interacting protein) regulate cell polarity and motility in neocortical subventricular and intermediate zones during radial migration.
AUTHOR: Nagano T.; Morikubo S.; Sato M.
CORPORATE SOURCE: M. Sato, Div. of Cell Biol. and Neuroscience, Dept. Morphological Physiological S., University of Fukui, Matsuoka, Fukui 910-1193, Japan. makosato@fmsrsa.fukui-med.ac.jp
SOURCE: Journal of Neuroscience, (27 Oct 2004) Vol. 24, No. 43, pp. 9648-9657.
Refs: 32
ISSN: 0270-6474 CODEN: JNRSDS
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20041119
Last Updated on STN: 20041119

L3 ANSWER 10 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI **Filamin A**, the Arp2/3 complex, and the morphology and function of cortical actin filaments in human melanoma cells.
AB The Arp2/3 complex and **filamin A** (FLNa) branch actin filaments. To define the role of these actin-binding proteins in cellular actin architecture, we compared the morphology of FLNa-deficient human melanoma (M2) cells and three stable derivatives of these cells expressing normal FLNa concentrations. All the cell lines contain similar amounts of the Arp2/3 complex. Serum **addition** causes serum-starved M2 cells to extend flat protrusions transiently; thereafter, the protrusions turn into spherical blebs and the cells do not crawl. The short-lived lamellae of M2 cells contain a dense mat of long actin filaments in contrast to a more three-dimensional orthogonal network of shorter actin filaments in lamellae of identically treated FLNa-expressing cells capable of translational locomotion. FLNa-specific antibodies localize throughout the leading lamellae of these cells at junctions between orthogonally intersecting actin filaments. Arp2/3 complex-specific antibodies stain diffusely and label a few, although not the same, actin filament overlap sites as FLNa antibody. We conclude that FLNa is essential in cells that express it for stabilizing orthogonal actin networks suitable for locomotion. Contrary to some proposals, Arp2/3 complex-mediated branching of actin alone is insufficient for establishing an orthogonal actin organization or maintaining mechanical stability at the leading edge.
ACCESSION NUMBER: 2002132328 EMBASE
TITLE: **Filamin A**, the Arp2/3 complex, and the morphology and function of cortical actin filaments in human melanoma cells.
AUTHOR: Flanagan L.A.; Chou J.; Falet H.; Neujahr R.; Hartwig J.H.; Stossel T.P.
CORPORATE SOURCE: T.P. Stossel, Hematology Division, Brigham and Women's Hospital, LMRC 301, 221 Longwood Ave., Boston, MA 02115, United States. tstossel@rics.bwh.harvard.edu
SOURCE: Journal of Cell Biology, (29 Oct 2001) Vol. 155, No. 3, pp. 511-517.
Refs: 23
ISSN: 0021-9525 CODEN: JCLBA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20020425
Last Updated on STN: 20020425

L3 ANSWER 11 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI [A proposal for a molecular genetic classification of the malformations of the nervous system].
PROPUESTA PARA UNA CLASIFICACION GENETICA MOLECULAR DE LAS MALFORMACIONES DEL SISTEMA NERVIOSO.

AB Objective. This proposal is a first attempt to incorporate the recent molecular genetic data that explains programming of development etiologically. Development. Traditional schemes of classifying nervous system malformations are based upon descriptive morphogenesis of anatomical processes of ontogeny, such as neurulation, neuroblast migration and axonal pathfinding; such anatomical schemes do not allow for the incorporation of multiple genetic etiologies that lead to the same anatomical result, such as holoprosencephaly or lissencephaly. A scheme based purely on genetic mutations also is impractical because several genes might be involved sequentially in a cascade, the same genes serve different functions at different stages and are involved in multiple organ systems. Some complex malformations result from several unrelated defective genes, again citing the example of holoprosencephaly. Finally, a pure genetic classification would be too inflexible to incorporate anatomical criteria and also acquired lesions of the fetal brain that lead to secondary focal dysgeneses. The basis for the proposed scheme is, therefore, disturbances in patterns of genetic expression: polarity gradients of the axes of the neural tube (e.g. upregulation or downregulation of genetic influences); segmentation (e.g. deletions of specific neuromeres; ectopic expression); mutations that cause change in cell lineage (e.g. dysplastic gangliocytoma of cerebellum; myofiber differentiation within brain); and specific genes or molecules that mediate neuroblast migration in its early (e.g. **filamin-1**), middle (e.g. LIS1; doublecortin) or late course (e.g. reelin; L1-CAM). Conclusions. The classification schemes that served so well throughout the 20th century no longer are adequate for the 21st century. The proposed scheme undoubtedly will undergo many future revisions, but it provides a starting point using currently available data.

ACCESSION NUMBER: 2001344078 EMBASE
TITLE: [A proposal for a molecular genetic classification of the malformations of the nervous system].
PROPUESTA PARA UNA CLASIFICACION GENETICA MOLECULAR DE LAS MALFORMACIONES DEL SISTEMA NERVIOSO.
AUTHOR: Sarnat H.B.
CORPORATE SOURCE: Dr. H.B. Sarnat, Children's Hosp./Reg. Med. Ctr., CH-49, 4800 Sand Point Way N.E., Seattle, WA 98105-0371, United States. hsarna@chmc.org
SOURCE: Revista de Neurologia, (2001) Vol. 33, No. 1, pp. 68-75.
Refs: 41
ISSN: 0210-0010 CODEN: RVNRAA
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 002 Physiology
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
022 Human Genetics
LANGUAGE: Spanish
SUMMARY LANGUAGE: English; Spanish; Portuguese
ENTRY DATE: Entered STN: 20011018
Last Updated on STN: 20011018

L3 ANSWER 12 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Periventricular heterotopia may result from radial glial fiber disruption.

AB Periventricular heterotopia (PVH) are collections of neurons and glia heterotopically located adjacent to the ventricles. The pathogenesis of periventricular heterotopia is believed to be a failure of cells to migrate from the ventricular zone. Mutations in **filamin-1** (FLN1) have recently been identified as a genetic defect that results in an X-linked dominant form of PVH. In **addition** to this X-linked form, PVH may be found sporadically or occasionally as part of other syndromes. The pathogenesis(es) of PVH has not been entirely

elucidated for patients with or without FLN1 mutation. In an attempt to better understand the pathogenesis of PVH, we examined 5 fetuses (gestational ages 21 to 34 wk), 3 females and 2 males, with PVH. Neuropathologic examination of these 5 fetuses revealed several to multiple periventricular nodules. No case showed the extensive periventricular heterotopia most commonly found in females with FLN1 mutations. By immunohistochemistry, neurofilament-positive cells were identified within the PVH in 3 of 5 cases and glial fibrillary acidic protein-positive cells were only found within the nodules of 3 cases. Surprisingly, small collections of CD68-positive macrophages were found at the base of the nodules in 4 of the 5 cases. Moreover, in all cases, the radial glia highlighted with vimentin, showed disorganization specifically around the nodules. These data suggest that at least one pathogenesis for PVH is a disruption of the radial glial organization, resulting in a failure of cells to migrate from the ventricular zone.

ACCESSION NUMBER: 2001331418 EMBASE
TITLE: Periventricular heterotopia may result from radial glial fiber disruption.
AUTHOR: Santi M.R.; Golden J.A.
CORPORATE SOURCE: Dr. J.A. Golden, Department of Pathology, Children's Hospital of Philadelphia, Abramson Research Center, 3400 Civic Center Blvd., Philadelphia, PA 19104, United States
SOURCE: Journal of Neuropathology and Experimental Neurology, (2001) Vol. 60, No. 9, pp. 856-862.
Refs: 32
ISSN: 0022-3069 CODEN: JNENAD
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20011004
Last Updated on STN: 20011004

L3 ANSWER 13 OF 14 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI **Cell migration** and cerebral cortical development.

AB This annotation describes the clinical and pathological features of several conditions believed to result from a primary defect in **cell migration** which include the lissencephalies, pachygria, polymicrogyrias, and focal cortical dysplasia. A variety of factors must be considered in pathogeneses, including cellular proliferation, cell death, post-migrational intracortical growth and development, axonogenesis and dendritogenesis. At least two distinct types of lissencephaly exist. Classic (also known as Type I) lissencephaly is the prototypic pattern being seen in autosomal dominant Miller-Dieker syndrome, in **addition** to autosomal recessive and X-linked forms. The Miller-Dieker syndrome locus (LIS-1) encodes the platelet activating factor acetylhydrolase-1, β1 subunit. The gene for an X-linked form of lissencephaly (XLIS) encodes a protein called doublecortin. Cobblestone (type II) lissencephaly is most commonly seen in patients with the Walker-Warburg syndrome, and also occurs in a group of disorders associated with congenital muscular dystrophy, including Finnish 'muscle-eye-brain' disease and Fukuyama muscular dystrophy. Controversy exits as to whether polymicrogyria is a malformation or a disruption of development. The answer is likely both, Polymicrogyria is believed to arise from defects occurring between 17 and 25 or 26 weeks gestation. Heterotopia can be sporadic, inherited as a simple Mendelian trait, or may be part of a more complex syndrome being characterized by collections of disorganized grey matter in inappropriate places. X-linked periventricular heterotopia syndrome is caused by mutations in **filamin-1**. In **addition** to those described above, many other syndromes show lissencephaly, pachygyria and polymicrogyria and many cases are not easily classified into any particular syndrome.

ACCESSION NUMBER: 2001139055 EMBASE

TITLE: **Cell migration** and cerebral cortical development.
AUTHOR: Golden J.A.
CORPORATE SOURCE: Dr. J.A. Golden, Department of Pathology, Abramson Research Center, Children's Hospital of Philadelphia, 3400 Civic Center Blvd, Philadelphia, PA 19104, United States. goldenj@mail.med.upenn.edu
SOURCE: Neuropathology and Applied Neurobiology, (2001) Vol. 27, No. 1, pp. 22-28.
Refs: 35
ISSN: 0305-1846 CODEN: NANEDL
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
021 Developmental Biology and Teratology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20010430
Last Updated on STN: 20010430

L3 ANSWER 14 OF 14 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Proteins controlling **cell migration** and cell death and their encoded DNAs, applicable in developing drugs for treating or suppressing cancer or tumor metastasis or as regulators of **cell migration** for transplantation.
AN 2003-268423 [26] WPIDS
AB WO2003018804 A UPAB: 20030428
NOVELTY - A DNA encoding:
(a) a protein containing an amino acid sequence of (II) with 1212 amino acids; or
(b) a protein based on the sequence (II) but with some amino acids deleted, substituted or added and having a function of controlling **cell migration** and cell death, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) a DNA containing all or a part of the base sequence with a sequence of (I) with 4364 base pairs, or its complementary strand;
(2) a DNA hybridizable with a DNA constituting the gene with a sequence of (I) under stringent conditions and encoding a protein with a function of controlling **cell migration** and cell death;
(3) a similar DNA encoding (a) a protein with an amino acid sequence of (IV) or (VI) of 964 or 1213 amino acids, respectively, or (b) a protein based on the sequence (IV) or (VI) but with some amino acids deleted, substituted or added and having a function of controlling **cell migration** and cell death;
(4) a DNA containing all or a part of the base sequence of (III) or (V) with 3785 or 4247 base pairs, respectively, or their complementary strand;
(5) a DNA hybridizable with a DNA constituting the gene with a sequence of (III) or (V) under stringent conditions and encoding a protein with a function of controlling **cell migration** and cell death;
(6) a protein containing an amino acid sequence of (II), (IV) or (VI);
(7) a protein based on the sequence of (II), (IV) or (VI) but with some amino acids deleted, substituted or added and having a function of controlling **cell migration** and cell death;
(8) a polypeptide containing a part of any of the proteins and having a function of controlling **cell migration** and cell death;
(9) a fused protein or fused peptide obtained by binding the protein or peptide with a marker protein and/or peptide;
(10) an antibody specifically binding to the protein or peptide;
(11) a recombinant protein or peptide binding specifically with the antibody;
(12) host cells containing an expression system to express the protein or peptide;

(13) a non-human animal with **deletion** of the gene function on the chromosome that encodes the protein or peptide;

(14) a non-human animal overexpressing the protein or peptide;

(15) screening substances that promote or inhibit the function of controlling **cell migration** and cell death by using any of the proteins, peptides, cell membranes expressing such proteins or peptides and a test substance;

(16) screening substances that can promote or inhibit expression of the protein or peptide by using any of the proteins and a test substance; or by using the non-human animal and the test substance;

(17) promoters or inhibitors thus screened; and

(18) cancer or tumor metastasis inhibitors or regulators of **cell migration** for transplantation therapy containing the (recombinant) proteins, (recombinant) peptides, screened promoters or screened inhibitors as active ingredient.

ACTIVITY - Cytostatic; Neuroprotective; Immunosuppressive.

No biological data given.

MECHANISM OF ACTION - None given.

USE - The proteins are for controlling **cell migration** and cell death, which is applicable in developing drugs for treating or suppressing cancer or tumor metastasis or as regulators of **cell migration** for transplantation therapy (claimed), and also for controlling the mobility and cell death of nerve cells, promoting decomposition of the actin-binding protein e.g. **filamin** -interacting protein in the treatment of preiventrilcular nodular heterotopia.

Dwg.0/4

ACCESSION NUMBER: 2003-268423 [26] WPIDS
DOC. NO. NON-CPI: N2003-213261
DOC. NO. CPI: C2003-070247
TITLE: Proteins controlling **cell migration** and cell death and their encoded DNAs, applicable in developing drugs for treating or suppressing cancer or tumor metastasis or as regulators of **cell migration** for transplantation.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): NAGANO, T; SATO, M
PATENT ASSIGNEE(S): (NAGA-I) NAGANO T; (SATO-I) SATO M; (NISC-N) JAPAN SCI & TECHNOLOGY CORP
COUNTRY COUNT: 3
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003018804	A1	20030306	(200326)*	JA	96
W: CA JP US					
US 2004229797	A1	20041118	(200477)		
JP 2003523653	X	20041209	(200481)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003018804	A1	WO 2002-JP7676	20020729
US 2004229797	A1 CIP of	WO 2002-JP7676	20020729
		US 2004-788793	20040227
JP 2003523653	X	WO 2002-JP7676	20020729
		JP 2003-523653	20020729

FILING DETAILS:

PATENT NO	KIND	PATENT NO
JP 2003523653	X Based on	WO 2003018804

PRIORITY APPLN. INFO: JP 2001-256910 20010827

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=> d his

     (FILE 'HOME' ENTERED AT 16:42:09 ON 15 APR 2005)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS'
     ENTERED AT 16:42:29 ON 15 APR 2005
L1          249 S FILAMIN A OR FILAMIN 1
L2           39 S L1 AND (CELL MIGRATION OR CELL DEATH CONTROL)
L3           14 S L2 AND (FRAGMENT OR VARIANT OR SUBSITUTION OR DELETION OR AD

=> s l2 and (modified amino acid sequence)
   3 FILES SEARCHED...
L4            0 L2 AND (MODIFIED AMINO ACID SEQUENCE)

=> s l3 and (encoding DNA)
   5 FILES SEARCHED...
L5            0 L3 AND (ENCODING DNA)

=> s l2 and DNA
L6           10 L2 AND DNA

=> d l6 ti abs ibib tot

L6   ANSWER 1 OF 10    MEDLINE on STN
TI   Interaction with BRCA2 suggests a role for filamin-1
     (hsFLNa) in DNA damage response.
AB   The BRCA2 tumor suppressor plays significant roles in DNA damage
     response.  The human actin binding protein filamin-1
     (hsFLNa, also known as ABP-280) participates in orthogonal actin network,
     cellular stress responses, signal transduction, and cell
     migration.  Through a yeast two-hybrid system, an in vitro binding
     assay, and in vivo co-immunoprecipitations, we identified an interaction
     between BRCA2 and hsFLNa.  The hsFLNa binding domain of BRCA2 was mapped
     to an internal conserved region, and the BRCA2-interacting domain of
     hsFLNa was mapped to its C terminus.  Although hsFLNa is known for its
     cytoplasmic functions in cell migration and signal
     transduction, some hsFLNa resides in the nucleus, raising the possibility
     that it participates in DNA damage response through a nuclear
     interaction with BRCA2.  Lack of hsFLNa renders a human melanoma cell line
     (M2) more sensitive to several genotoxic agents including gamma
     irradiation, bleomycin, and ultraviolet-c light.  These results suggest
     that BRCA2/hsFLNa interaction may serve to connect cytoskeletal signal
     transduction to DNA damage response pathways.
ACCESSION NUMBER:    2001698266     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 11602572
TITLE:               Interaction with BRCA2 suggests a role for filamin
                     -1 (hsFLNa) in DNA damage response.
AUTHOR:              Yuan Y; Shen Z
CORPORATE SOURCE:    Department of Molecular Genetics and Microbiology,
                     University of New Mexico School of Medicine, Albuquerque,
                     New Mexico 87131, USA.
CONTRACT NUMBER:     ES08353 (NIEHS)
SOURCE:              Journal of biological chemistry, (2001 Dec 21) 276 (51)
                     48318-24.  Electronic Publication: 2001-10-15.
                     Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200201
ENTRY DATE:          Entered STN: 20011218
                     Last Updated on STN: 20030105
                     Entered Medline: 20020131

L6   ANSWER 2 OF 10  USPATFULL on STN
TI     Specific markers for diabetes
AB     The present invention provides polypeptides which are correlated with
       pre-diabetes, diabetes or susceptibility to diabetes which can be used
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as markers for diagnosis of pre-diabetes, diabetes or a susceptibility or predisposition to develop diabetes. The invention also provides methods for the diagnosis of pre-diabetes, diabetes and/or the susceptibility to diabetes by obtaining a biological sample and detecting and/or measuring the increase of one or more polypeptides as disclosed herein. Screening methods relating to agonists and antagonists of the specific polypeptides disclosed herein are provided. Antibodies may also be raised against these polypeptide markers for the detection and/or treatment of diabetes. Proteins, protein fragments or peptides can be used for the treatment of diabetes or pre-diabetes.

ACCESSION NUMBER: 2005:87343 USPATFULL
TITLE: Specific markers for diabetes
INVENTOR(S): Kochan, Jarema Peter, Towaco, NJ, UNITED STATES
Martin, Mitchell Lee, Verona, NJ, UNITED STATES
Rosinski, James Andrew, Nutley, NJ, UNITED STATES
PATENT ASSIGNEE(S): Hoffmann-La Roche Inc., Nutley, NJ (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005074805	A1	20050407
APPLICATION INFO.:	US 2004-952459	A1	20040928 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-508699P	20031003 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOFFMANN-LA ROCHE INC., PATENT LAW DEPARTMENT, 340 KINGSLAND STREET, NUTLEY, NJ, 07110
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 2961

L6 ANSWER 3 OF 10 USPATFULL on STN
TI Proteins having effects of controlling **cell migration** and cell death
AB The present invention relates to a protein having effects of controlling **cell migration** and cell death of such as neurons and a **DNA** encoding the protein, and an object of the present invention is to provide control **cell migration** and/or cell death and a method for screening a promoter or an inhibitor of the effects of controlling **cell migration** and/or cell death with the use of proteins controlling the cell motility and cell death of neurons by interacting particularly with an actin-binding protein and promoting the degradation of the actin-binding protein and the **DNA** encoding the proteins. S-FILIP, L-FILIP and h-FILIP cDNAs, interacting with an actin-binding protein **Filamin 1**, and negatively controlling **cell migration** by promoting the degradation of the **Filamin 1**, and involved in the control of the cell death, were isolated and the full base sequences and amino acid sequences thereof were determined.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:292708 USPATFULL
TITLE: Proteins having effects of controlling **cell migration** and cell death
INVENTOR(S): Sato, Makoto, Fukui-shi, JAPAN
Nagano, Takashi, Sakai-gun, JAPAN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004229797	A1	20041118
APPLICATION INFO.:	US 2004-788793	A1	20040227 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2002-JP7676, filed on 29 Jul 2002, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	JP 2001-256910	20010827

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: THOMAS J. KOWALSKI, Esq., c/o FROMMER LAWRENCE & HAUG LLP, 745 Fifth Avenue, New York, NY, 10151
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 2861
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 10 USPATFULL on STN
TI Specific markers for pancreatic cancer
AB The present invention provides polypeptides which are up- or down-regulated in pancreatic cancer and which can be used as markers for diagnosis of pancreatic cancer. The invention also provides an in vitro method for the diagnosis of pancreatic cancer and/or the susceptibility to pancreatic cancer comprising the steps of a) obtaining a biological sample; and b) detecting and/or measuring the increase of one or more polypeptides as disclosed herein. Furthermore, screening methods relating to inhibitors and antagonists of the specific polypeptides disclosed herein are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:280272 USPATFULL
TITLE: Specific markers for pancreatic cancer
INVENTOR(S): Chen, Jie, Beijing, CHINA
Hu, Liping, Beijing, CHINA
Liu, Tong Hua, Beijing, CHINA
Lu, Zhao Hui, Beijing, CHINA
Shen, Yan, Beijing, CHINA

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004219572	A1	20041104	
APPLICATION INFO.:	US 2003-733969	A1	20031211	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	EP 2002-28058	20021217
	EP 2003-25237	20031105

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOFFMANN-LA ROCHE INC., PATENT LAW DEPARTMENT, 340 KINGSLAND STREET, NUTLEY, NJ, 07110
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
LINE COUNT: 8167
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 10 USPATFULL on STN
TI Modulators of angiogenesis
AB The present invention relates to regulation of angiogenesis. More particularly, the present invention is directed to nucleic acids encoding "angiogenesis regulatory proteins and nucleic acids" which are involved in modulation of angiogenesis. The invention further relates to methods for identifying and using agents, including small organic molecules, antibodies, peptides, cyclic peptides, nucleic acids, antisense nucleic acids, RNAi, and ribozymes, that modulate angiogenesis via modulation of angiogenesis regulatory proteins and nucleic acids; as well as to the use of expression profiles and compositions in diagnosis and therapy of diseases related to angiogenesis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:70006 USPATFULL
TITLE: Modulators of angiogenesis

INVENTOR(S): Lorens, James B., Portola Valley, CA, UNITED STATES
Xu, Weiduan, San Francisco, CA, UNITED STATES
Bogenberger, Jakob, San Francisco, CA, UNITED STATES
Holland, Sacha, San Francisco, CA, UNITED STATES
PATENT ASSIGNEE(S): Rigel Pharmaceuticals, Incorporated, South San Francisco, CA, UNITED STATES, 94080 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004053233	A1	20040318	
APPLICATION INFO.:	US 2002-231956	A1	20020830	(10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 37
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 2914
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 6 OF 10 USPATFULL on STN
TI GIPs, a family of polypeptides with transcription factor activity that interact with goodpasture antigen binding protein
AB The present invention provides isolated GPBP-interacting 90 and 130 kDa polypeptides, and portions thereof (GIP90/130 polypeptides), antibodies to the GIP90/130 polypeptides, and pharmaceutical compositions thereof. The present invention also provides isolated GIP90/130 nucleic acid sequences, expression vectors comprising the nucleic acid sequences, and host cells transfected with the expression vectors. The invention further provides methods for detecting the GIP90/130 polypeptides or nucleic acid sequences, methods for inhibiting interactions between GPBP and GIP90/130 polypeptides, between pol k76 and GIP90/130 polypeptides or aggregation of GIP90/130 polypeptides, and methods for treating patients with autoimmune disorders or cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:158941 USPATFULL
TITLE: GIPs, a family of polypeptides with transcription factor activity that interact with goodpasture antigen binding protein
INVENTOR(S): Saus, Juan, Valencia, SPAIN
Revert-Ros, Francisco, Valencia, SPAIN

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003108554	A1	20030612	
APPLICATION INFO.:	US 2002-309851	A1	20021204	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-338287P	20011207	(60)
	US 2002-382004P	20020520	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606
NUMBER OF CLAIMS: 33
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 3697
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 10 USPATFULL on STN
TI Diagnosis and treatment of medical conditions associated with defective NFkappa B(NF-kappaB) activation
AB Incontinentia Pigmenti (IP) is a neurocutaneous genodermatosis that segregates as an X-linked dominant disorder with a high probability of

prenatal male lethality. A locus in Xq28 containing NF-κB Essential Modulator, a gene product involved in the activation of NF-kB and central to many pro-inflammatory and apoptotic pathways, contains mutations in the majority of cases of IP. Disclosed are methods, compositions and kits directed to a defect in a NF-κB related disease such as IP.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:44745 USPATFULL

TITLE: Diagnosis and treatment of medical conditions associated with defective NFkappa B(NF-kappaB) activation

INVENTOR(S): Kenwrick, Sue J., Cambridge, UNITED KINGDOM
Woffendin, Hayley, Cambridge, UNITED KINGDOM
Munnich, Arnold, Paris, FRANCE
Smahi, Asmae, Saint Ouen, FRANCE
Israel, Alain, Paris, FRANCE
Poustka, Annemarie, Heidelberg, GERMANY, FEDERAL REPUBLIC OF
Heiss, Nina, Heidelberg, GERMANY, FEDERAL REPUBLIC OF
D'Urso, Michele, Napoli, ITALY
Lewis, Richard Alan, Houston, TX, UNITED STATES
Nelson, David L., Houston, TX, UNITED STATES
Aradhya, Swaroop, Houston, TX, UNITED STATES
Levy, Moise, Houston, TX, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003032055	A1	20030213	
	US 6824972	B2	20041130	
APPLICATION INFO.:	US 2001-863049	A1	20010522	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-206223P	20000522 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FULBRIGHT & JAWORSKI, LLP, 1301 MCKINNEY, SUITE 5100, HOUSTON, TX, 77010-3095
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 3161
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 8 OF 10 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Interaction with BRCA2 Suggests a Role for **Filamin-1** (hsFLNa) in **DNA** Damage Response.

AB The BRCA2 tumor suppressor plays significant roles in **DNA** damage response. The human actin binding protein **filamin-1** (hsFLNa, also known as ABP-280) participates in orthogonal actin network, cellular stress responses, signal transduction, and **cell migration**. Through a yeast two-hybrid system, an in vitro binding assay, and in vivo co-immunoprecipitations, we identified an interaction between BRCA2 and hsFLNa. The hsFLNa binding domain of BRCA2 was mapped to an internal conserved region, and the BRCA2-interacting domain of hsFLNa was mapped to its C terminus. Although hsFLNa is known for its cytoplasmic functions in **cell migration** and signal transduction, some hsFLNa resides in the nucleus, raising the possibility that it participates in **DNA** damage response through a nuclear interaction with BRCA2. Lack of hsFLNa renders a human melanoma cell line (M2) more sensitive to several genotoxic agents including γ irradiation, bleomycin, and ultraviolet-c light. These results suggest that BRCA2/hsFLNa interaction may serve to connect cytoskeletal signal transduction to **DNA** damage response pathways.

ACCESSION NUMBER: 2003451631 EMBASE

TITLE: Interaction with BRCA2 Suggests a Role for **Filamin**

-1 (hsFLNa) in **DNA** Damage Response.
AUTHOR: Yuan Y.; Shen Z.
CORPORATE SOURCE: Z. Shen, Dept. Molec. Genet. and Microbiol., Univ. of New Mexico Sch. of Medicine, 915 Camino de Salud, NE, Albuquerque, NM 87131, United States. zshen@salud.unm.edu
SOURCE: Journal of Biological Chemistry, (21 Dec 2001) Vol. 276, No. 51, pp. 48318-48324.
Refs: 47
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20031211
Last Updated on STN: 20031211

L6 ANSWER 9 OF 10 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Mutations in the X-linked **filamin 1** gene cause periventricular nodular heterotopia in males as well as in females.
AB Periventricular heterotopia (PH) is a human neuronal migration disorder in which many neurons destined for the cerebral cortex fail to migrate. Previous analysis showed heterozygous mutations in the X-linked gene **filamin 1** (FLN1), but examined only the first six (of 48) coding exons of the gene and hence did not assess the incidence and functional consequences of FLN1 mutations. Here we perform single-strand conformation polymorphism (SSCP) analysis of FLN1 throughout its entire coding region in six PH pedigrees, 31 sporadic female PH patients and 24 sporadic male PH patients. We detected FLN1 mutations by SSCP in 83% of PH pedigrees and 19% of sporadic females with PH. Moreover, no PH females (0/7 tested) with atypical radiographic features showed FLN1 mutations, suggesting that other genes may cause atypical PH. Surprisingly, 2/24 males analyzed with PH (9%) also carried FLN1 mutations. Whereas FLN1 mutations in PH pedigrees caused severe predicted loss of FLN1 protein function, both male FLN1 mutations were consistent with partial loss of function of the protein. Moreover, sporadic female FLN1 mutations associated with PH appear to cause either severe or partial loss of function. Neither male could be shown to be mosaic for the FLN1 mutation in peripheral blood lymphocytes, suggesting that some neurons in the intact cortex of PH males may be mutant for FLN1 but migrate adequately. These results demonstrate the sensitivity and specificity of **DNA** testing for FLN1 mutations and have important functional implications for models of FLN1 protein function in neuronal migration.
ACCESSION NUMBER: 2001338315 EMBASE
TITLE: Mutations in the X-linked **filamin 1** gene cause periventricular nodular heterotopia in males as well as in females.
AUTHOR: Sheen V.L.; Dixon P.H.; Fox J.W.; Hong S.E.; Kinton L.; Sisodiya S.M.; Duncan J.S.; Dubeau F.; Scheffer I.E.; Schachter S.C.; Wilner A.; Henchy R.; Crino P.; Kamuro K.; DiMario F.; Berg M.; Kuzniecky R.; Cole A.J.; Bromfield E.; Biber M.; Schomer D.; Wheless J.; Silver K.; Mochida G.H.; Berkovic S.F.; Andermann F.; Andermann E.; Dobyns W.B.; Wood N.W.; Walsh C.A.
CORPORATE SOURCE: C.A. Walsh, Harvard Institutes of Medicine, Department of Neurology, Beth Israel Deaconess Medical Center, 77 Avenue Louis Pasteur, Boston, MA 02115, United States. cwalsh@caregroup.harvard.edu
SOURCE: Human Molecular Genetics, (15 Aug 2001) Vol. 10, No. 17, pp. 1775-1783.
Refs: 31
ISSN: 0964-6906 CODEN: HMGEE5
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery

014 Radiology
022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20011011
Last Updated on STN: 20011011

L6 ANSWER 10 OF 10 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Proteins controlling **cell migration** and cell death and their encoded DNAs, applicable in developing drugs for treating or suppressing cancer or tumor metastasis or as regulators of **cell migration** for transplantation.
AN 2003-268423 [26] WPIDS
AB WO2003018804 A UPAB: 20030428
NOVELTY - A **DNA** encoding:
(a) a protein containing an amino acid sequence of (II) with 1212 amino acids; or
(b) a protein based on the sequence (II) but with some amino acids deleted, substituted or added and having a function of controlling **cell migration** and cell death, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) a **DNA** containing all or a part of the base sequence with a sequence of (I) with 4364 base pairs, or its complementary strand;
(2) a **DNA** hybridizable with a **DNA** constituting the gene with a sequence of (I) under stringent conditions and encoding a protein with a function of controlling **cell migration** and cell death;
(3) a similar **DNA** encoding (a) a protein with an amino acid sequence of (IV) or (VI) of 964 or 1213 amino acids, respectively, or (b) a protein based on the sequence (IV) or (VI) but with some amino acids deleted, substituted or added and having a function of controlling **cell migration** and cell death;
(4) a **DNA** containing all or a part of the base sequence of (III) or (V) with 3785 or 4247 base pairs, respectively, or their complementary strand;
(5) a **DNA** hybridizable with a **DNA** constituting the gene with a sequence of (III) or (V) under stringent conditions and encoding a protein with a function of controlling **cell migration** and cell death;
(6) a protein containing an amino acid sequence of (II), (IV) or (VI);
(7) a protein based on the sequence of (II), (IV) or (VI) but with some amino acids deleted, substituted or added and having a function of controlling **cell migration** and cell death;
(8) a polypeptide containing a part of any of the proteins and having a function of controlling **cell migration** and cell death;
(9) a fused protein or fused peptide obtained by binding the protein or peptide with a marker protein and/or peptide;
(10) an antibody specifically binding to the protein or peptide;
(11) a recombinant protein or peptide binding specifically with the antibody;
(12) host cells containing an expression system to express the protein or peptide;
(13) a non-human animal with deletion of the gene function on the chromosome that encodes the protein or peptide;
(14) a non-human animal overexpressing the protein or peptide;
(15) screening substances that promote or inhibit the function of controlling **cell migration** and cell death by using any of the proteins, peptides, cell membranes expressing such proteins or peptides and a test substance;
(16) screening substances that can promote or inhibit expression of the protein or peptide by using any of the proteins and a test substance; or by using the non-human animal and the test substance;
(17) promoters or inhibitors thus screened; and
(18) cancer or tumor metastasis inhibitors or regulators of **cell migration** for transplantation therapy containing

the (recombinant) proteins, (recombinant) peptides, screened promoters or screened inhibitors as active ingredient.

ACTIVITY - Cytostatic; Neuroprotective; Immunosuppressive.
No biological data given.
MECHANISM OF ACTION - None given.
USE - The proteins are for controlling **cell migration** and cell death, which is applicable in developing drugs for treating or suppressing cancer or tumor metastasis or as regulators of **cell migration** for transplantation therapy (claimed), and also for controlling the mobility and cell death of nerve cells, promoting decomposition of the actin-binding protein e.g. **filamin** -interacting protein in the treatment of preiventrilcular nodular heterotopia.
Dwg.0/4

ACCESSION NUMBER: 2003-268423 [26] WPIDS
DOC. NO. NON-CPI: N2003-213261
DOC. NO. CPI: C2003-070247
TITLE: Proteins controlling **cell migration** and cell death and their encoded DNAs, applicable in developing drugs for treating or suppressing cancer or tumor metastasis or as regulators of **cell migration** for transplantation.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): NAGANO, T; SATO, M
PATENT ASSIGNEE(S): (NAGA-I) NAGANO T; (SATO-I) SATO M; (NISC-N) JAPAN SCI & TECHNOLOGY CORP
COUNTRY COUNT: 3
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003018804	A1	20030306	(200326)*	JA	96
W: CA JP US					
US 2004229797	A1	20041118	(200477)		
JP 2003523653	X	20041209	(200481)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003018804	A1	WO 2002-JP7676	20020729
US 2004229797	A1 CIP of	WO 2002-JP7676	20020729
		US 2004-788793	20040227
JP 2003523653	X	WO 2002-JP7676	20020729
		JP 2003-523653	20020729

FILING DETAILS:

PATENT NO	KIND	PATENT NO
JP 2003523653	X Based on	WO 2003018804

PRIORITY APPLN. INFO: JP 2001-256910 20010827

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FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS'
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L1 249 S FILAMIN A OR FILAMIN 1
L2 39 S L1 AND (CELL MIGRATION OR CELL DEATH CONTROL)
L3 14 S L2 AND (FRAGMENT OR VARIANT OR SUBSITUTION OR DELETION OR AD
L4 0 S L2 AND (MODIFIED AMINO ACID SEQUENCE)
L5 0 S L3 AND (ENCODING DNA)
L6 10 S L2 AND DNA

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=> e nagano,T/au
E1            1     NAGANO ZENJI/AU
E2            4     NAGANO ZENTARO/AU
E3            0 --> NAGANO,T/AU
E4            2     NAGANOBE MIKIO/AU
E5           22     NAGANOBU K/AU
E6           16     NAGANOBU KIYOKAZU/AU
E7           14     NAGANOBU M/AU
E8           18     NAGANOBU MIKIO/AU
E9            2     NAGANOH M/AU
E10           4     NAGANOH MASATAKE/AU
E11           2     NAGANOMA H/AU
E12          71     NAGANOMA HIROSHI/AU

=> e sato, m/au
E1            2     SATO ZIN/AU
E2            2     SATO ZYOUJI/AU
E3            0 --> SATO, M/AU
E4            1     SATO2 SHINSUKE/AU
E5            1     SATOA AKIRA/AU
E6            1     SATOA M/AU
E7            1     SATOASA AKIRA/AU
E8            1     SATOB S/AU
E9            1     SATOBAYASHI H/AU
E10           1     SATOBI YUICHI/AU
E11           1     SATOBUKA FUMIHIKO/AU
E12           3     SATOBUKA YOSHIFUMI/AU

=> s l6 and hybridize
L7            3 L6 AND HYBRIDIZE

=> d l7 ti abs ibib tot

L7   ANSWER 1 OF 3  USPATFULL on STN
TI     Modulators of angiogenesis
AB     The present invention relates to regulation of angiogenesis. More
       particularly, the present invention is directed to nucleic acids
       encoding "angiogenesis regulatory proteins and nucleic acids" which are
       involved in modulation of angiogenesis. The invention further relates to
       methods for identifying and using agents, including small organic
       molecules, antibodies, peptides, cyclic peptides, nucleic acids,
       antisense nucleic acids, RNAi, and ribozymes, that modulate angiogenesis
       via modulation of angiogenesis regulatory proteins and nucleic acids; as
       well as to the use of expression profiles and compositions in diagnosis
       and therapy of diseases related to angiogenesis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:        2004:70006  USPATFULL
TITLE:                   Modulators of angiogenesis
INVENTOR(S):             Lorens, James B., Portola Valley, CA, UNITED STATES
                         Xu, Weiduan, San Francisco, CA, UNITED STATES
                         Bogenberger, Jakob, San Francisco, CA, UNITED STATES
                         Holland, Sacha, San Francisco, CA, UNITED STATES
PATENT ASSIGNEE(S):      Rigel Pharmaceuticals, Incorporated, South San
                         Francisco, CA, UNITED STATES, 94080 (U.S. corporation)

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2004053233      A1   20040318
APPLICATION INFO.:       US 2002-231956     A1   20020830  (10)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO
                         CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS:        37
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      2 Drawing Page(s)
LINE COUNT:              2914
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 2 OF 3 USPATFULL on STN

TI GIPs, a family of polypeptides with transcription factor activity that interact with goodpasture antigen binding protein

AB The present invention provides isolated GPBP-interacting 90 and 130 kDa polypeptides, and portions thereof (GIP90/130 polypeptides), antibodies to the GIP90/130 polypeptides, and pharmaceutical compositions thereof. The present invention also provides isolated GIP90/130 nucleic acid sequences, expression vectors comprising the nucleic acid sequences, and host cells transfected with the expression vectors. The invention further provides methods for detecting the GIP90/130 polypeptides or nucleic acid sequences, methods for inhibiting interactions between GPBP and GIP90/130 polypeptides, between pol k76 and GIP90/130 polypeptides or aggregation of GIP90/130 polypeptides, and methods for treating patients with autoimmune disorders or cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:158941 USPATFULL

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003108554	A1	20030612
APPLICATION INFO.:	US 2002-309851	A1	20021204 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-338287P	20011207 (60)
	US 2002-382004P	20020520 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606
NUMBER OF CLAIMS: 33
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 3697

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 3 OF 3 USPATFULL on STN

TI Diagnosis and treatment of medical conditions associated with defective NFkappa B(NF-kappaB) activation

AB Incontinentia Pigmenti (IP) is a neurocutaneous genodermatosis that segregates as an X-linked dominant disorder with a high probability of prenatal male lethality. A locus in Xq28 containing NF-κB Essential Modulator, a gene product involved in the activation of NF-kB and central to many pro-inflammatory and apoptotic pathways, contains mutations in the majority of cases of IP. Disclosed are methods, compositions and kits directed to a defect in a NF-κB related disease such as IP.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:44745 USPATFULL
TITLE: Diagnosis and treatment of medical conditions associated with defective NFkappa B(NF-kappaB) activation
INVENTOR(S): Kenwrick, Sue J., Cambridge, UNITED KINGDOM
Woffendin, Hayley, Cambridge, UNITED KINGDOM
Munnich, Arnold, Paris, FRANCE
Smahi, Asmae, Saint Ouen, FRANCE
Israel, Alain, Paris, FRANCE
Poustka, Annemarie, Heidelberg, GERMANY, FEDERAL REPUBLIC OF

Heiss, Nina, Heidelberg, GERMANY, FEDERAL REPUBLIC OF
D'Urso, Michele, Napoli, ITALY
Lewis, Richard Alan, Houston, TX, UNITED STATES
Nelson, David L., Houston, TX, UNITED STATES
Aradhya, Swaroop, Houston, TX, UNITED STATES
Levy, Moise, Houston, TX, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003032055	A1	20030213
	US 6824972	B2	20041130
APPLICATION INFO.:	US 2001-863049	A1	20010522 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-206223P	20000522 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FULBRIGHT & JAWORSKI, LLP, 1301 MCKINNEY, SUITE 5100, HOUSTON, TX, 77010-3095
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 3161
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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 NEWS  2          "Ask CAS" for self-help around the clock
 NEWS  3  FEB 25  CA/CAPLUS - Russian Agency for Patents and Trademarks
                  (ROSPATENT) added to list of core patent offices covered
 NEWS  4  FEB 28  PATDPAFULL - New display fields provide for legal status
                  data from INPADOC
 NEWS  5  FEB 28  BABS - Current-awareness alerts (SDIs) available
 NEWS  6  FEB 28  MEDLINE/LMEDLINE reloaded
 NEWS  7  MAR 02  GBFULL: New full-text patent database on STN
 NEWS  8  MAR 03  REGISTRY/ZREGISTRY - Sequence annotations enhanced
 NEWS  9  MAR 03  MEDLINE file segment of TOXCENTER reloaded
 NEWS  10 MAR 22  KOREAPAT now updated monthly; patent information enhanced
 NEWS  11 MAR 22  Original IDE display format returns to REGISTRY/ZREGISTRY
 NEWS  12 MAR 22  PATDPASPC - New patent database available
 NEWS  13 MAR 22  REGISTRY/ZREGISTRY enhanced with experimental property tags
 NEWS  14 APR 04  EPFULL enhanced with additional patent information and new
                  fields
 NEWS  15 APR 04  EMBASE - Database reloaded and enhanced

 NEWS EXPRESS  JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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=> file medline, uspatful, dgene, embase, wpids, fsta, jicst
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                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 0.21       0.21

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FILE 'USPATFULL' ENTERED AT 16:42:29 ON 15 APR 2005
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L1   249 FILAMIN A OR FILAMIN 1